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CUMULATIVE CHLORINE POISONING ON BOARD A SUBMARINE

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CUMULATIVE CHLORINE POISONING ON BOARD A SUBMARINE
[Intossicazione collecttiva da gas cloro a bordo di un sommergibile]

by

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Statement of the Situation

On board the submarine "Nereide" during a war mission, the escape of chlorine gas took place because of the seepage of seawater into the batteries. This happened through an inadvertant opening of the exhaust valve of the batteries that gave rise to the entry of seawater which poured into the stringer around the coaming of the battery of the stern and crossed the vents and the cracks in the deck putty and filtered into several of the batteries' elements.

Consequently, the chlorides of the seawater, principally sodium chloride, reacted with the sulfuric acid contained in the storage batteries in the form of a watery solution developing, together with other secondary products, sodium bisulfate, hydrochloric acid which remained in solution, and chlorine in the gaseous state.

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The commander of the submarine, warned of the presence of the gas, surfaced and ordered the opening of all portholes for a complete ventilation of the compartments and the use of masks for the guard personnel of the thermic engine and for those who had to dry the water in the stringer of the auxiliary engine system.

It is useful to make several observations in order to be able to explain a series of unhealthy phenomena exhibited by a good part of the crew:

1) It came to my attention that, at an early time, the electronics personnel who were in the auxiliary engine room to dry the water in the stringer, gave warning of a slightly bitter odor diffused in the atmosphere; this was said to have little importance since they attributed it to the normal exhaust that the various engines commonly produced. Because the odor slowly became more pronounced, so much so that the personnel in the contiguous rooms (thermic engines and control room) were also warned, it began to interfere with respiration. Thus it was that the commander ordered a rapid surfacing, the opening of all the portholes, and the use of gas masks; all of this happened, according to the information gathered and the statements of the same personnel, about a half-hour after the very first traces of the gas (bitter odor) were detected, and about a quarter of an hour after the crew had begun to manifest the first annoying respiratory symptoms (cough, sense of constriction in the thorax, difficulty in breathing).

Nevertheless, for about a quarter of an hour, the personnel breathed air poisoned to a concentration of chlorine equal to or higher than 100 mmgr. per m^3 (the maximum limit that this can be withstood without damage by a normal man is 1 min).

2) Gas masks were applied only by those in the thermic engine room and in the auxiliary engine room.

3) The inquiry showed that some of the personnel, eluding the watch of their superiors, wore the masks for only a few minutes and then, feeling uncomfortable, removed them while continuing guard duty in the same rooms.

Medical Observations Concerning the Stricken

Immediately upon the return of the submarine to its base, all of the crew underwent medical examinations. Almost the entire crew of 47 persons referred to having sensed, at sea, a peculiar, bitter odor and to having had several coughing spells; however, apart from the spells, all problems ceased with the surfacing and the ventilation of the compartments involved.

On the other hand, 26 men had experienced a much greater poisoning, the symptoms of which were manifested at sea: strong constriction in the thorax, difficulty in breathing with a sense of suffocation, spasmodic coughing, heaviness in the head, dizziness, nausea, attempts at vomiting and, in some cases, actual vomiting. On their arrival at the base, they were complaining of a variety of disturbances, consisting of a strong tickle in the throat, abundant salivation, a bitter taste in the mouth, headaches, a retrosternal pain, asthenia, gastric burning, nausea, lack of appetite, stomach cramps. The tongue was covered with a dense yellowish coating. There was no fever, and no further symptoms appeared in objective examination.

There was a certain interest in knowing in which compartments of the submarine the 26 men who were particularly affected by the gas were, since this could explain the different states of severity exhibited by the individuals. The results were:

Six men had been in the auxiliary engine room,

Five in the thermic engine room,

Five in the control room, two of whom, however, had lingered for several minutes, without gas masks, in the auxiliary engine room,

Five in the forward torpedo room,

Five in the rear torpedo room.

The personnel who were most effected by the poison and who exhibited more long-run disturbances, both subjective and objective, were those in the auxiliary engine room where the gas was generated and in the thermic engine room which is contiguous to the former and is not very well ventilated, that is to say the electricians and the mechanics. The personnel who were located in the stern and the prow and who were without masks were reached by the gas through its rapid diffusion.

The 26 individuals were the objects of daily surveillance and ideal care, and frequently underwent medical examinations, the results of which can be summarized by the following clinical symptoms:

Irritable cough: 2 cases

Lack of appetite and nausea: 3 cases

Stomach cramps and gastric burning: 3 cases

Asthenia: 7 cases

Vertigo: 2 cases

Retrosternal pain: 21 cases

Coated tongue: 15 cases

Irritation in the throat: 9 cases

Bitter taste in mouth: 21 cases

Headaches: 16 cases

Abundant salivation: 12 cases

Palpable spleen: 1 case

Slight jaundice of the sclera: 2 cases

Liver painful to the touch: 4 cases

Palpable and painful liver: 8 cases

Acute bronchial catarrh: 3 cases

For the beginning, the progress, and the duration of each clinical symptom, see the attached table.

Table

Results of the medical exam

	Day after arrival	4th day of observation	7th day	11th day	21st day	27th day	50th day
Irritable cough	2	1	-	-	-	-	-
Lack of appetite and nausea	3	1	-	-	-	-	-
Stomach cramps and gastric burning	3	1	-	-	-	-	-
Asthenia	7	-	-	1	-	-	-
Vertigo	2	1	1	1	1	-	-
Retrosternal pain	13	5	-	-	-	-	-
Coated tongue	21	-	-	-	-	-	-
Irritation in the throat	15	5	2	-	-	-	-
Bitter taste in mouth	9	8	2	-	-	-	-
Headaches	16	8	2	1	-	-	-
Abundant salivation	12	-	-	-	-	-	-
Palpable spleen	-	1	1	-	-	-	-
Slight jaundice of the sclera	-	2	-	-	-	-	-
Liver painful to the touch	-	4	2	1	1	-	-
Palpable and painful liver	8	7	4	4	4	4	4
Acute bronchial catarrh	-	-	3	3	2	2	-

On the fourth day of treatment and observation, 11 persons who still exhibited only light headaches, light irritation in the throat, and light retrosternal pain were able to report for duty while continuing symptomatic treatment. On the twentieth day, all the personnel were able to resume normal activities on board the submarine -- with the exception of one individual who still exhibited acute diffused bronchial catarrh in evolutive form.

After a 28-day stay at the base, the submarine left for a new mission; upon reentry, about 50 days after the accident, another test was carried out (see the table).

Dietary and Therapeutic Measures Adopted

Immediately upon their arrival at the base, I administered egg albumin and burnt magnesium for the acute gastric symptoms.

I prescribed mouthwashes and gomenol oil for the respiratory tract. The patients were put on a milk diet and were to have absolute rest.

On the fourth day, a part of the stricken crew members were able to take food again and resume normal activities. For the remaining members, I prescribed a 15-day light diet based on milk, butter, and greens, and rest in the open air.

As detoxifying therapy in general and for the liver in particular, rhubarb, two podophyllin compresses a day, 10 grams of magnesiumsulfate daily in the morning on an empty stomach, and intravenous urotropine were administered.

For the acute bronchial inflamation, there was bed rest, 5 cc of calcium gluconate a day, syrup of snakeroot, compresses of guaiacol carbonate, and thioseptal [sic].

Clinical Considerations Concerning Several Symptoms Observed in the Poisoned Patients

1) On the fourth day of observation, I wanted to investigate the liver conditions in those poisoned and, using the common semeiotic technique, I obtained the following results:

In seven patients, a palpable and painful liver;

In one patient, a barely palpable and painful liver;

In four patients, a liver painful to the touch with Murphy's maneuver.

Characteristics of the hepatomegaly: The liver extending about 5 cm from the costal arch at deep inspiration; in the mammillary, a uniformly smooth surface with a sharp, cutting edge easily turned up under the fingers with a soft, elastic consistency; upper limit to the sixth rib at the mammillary.

Just then, I was systematically examining the liver of the submariners of the fifth group and I had determined that about 10% of the personnel were exhibiting hepatomegaly of which the principal characteristics were: a soft, elastic consistency and difficulty in regressing with time.

I still had not directed my research to the submarine in question. When I did, I found that another two persons from the submarine, who had not been poisoned by the chlorine, were exhibiting hepatomegaly. Therefore, among the personnel of the submarine, there were ten individuals with enlarged livers, eight of whom had been attacked by the chlorine and two who had not. I then began to wonder whether some cases of hepatomegaly exhibited by the poisoned individuals were due not to chlorine but instead were the expression of a slow, gradual poisoning to which the personnel of the submarine were subjected.

From subsequent medical examinations carried out on the personnel attacked by the chlorine (see the table), it seemed that hepatomegaly was no longer traceable in four of the eight subjects about 11 days after the poisoning.

On the other hand, in the other four subjects, it remained unchanged in a test that I was able to administer about 50 days after the accident; I was able to establish the same persistence in the two hepatomegaly cases that were not caused by the chlorine.

I nevertheless feel that I can conclude that:

a) The hepatic pain on palpitation, manifested by itself in four individuals and unaccompanied by obvious hepatomegaly, was due to a form of initial, acute hepatic congestion from the absorption of chlorine.

b) The hepatomegaly exhibited by the four subjects in whom it disappeared after a few days was due to a more advanced state of gas chlorine congestion, which regressed at the same rate as the poisoning of the organisms attacked lessened.

c) The hepatomegaly exhibited by the four subjects in whom it remained unchanged throughout all of the medical examinations, while also able to have an acute component due to the absorption of chlorine, was, on the other hand, an expression of a long-term hepatic change that is one of the diseased forms which the slow poisoning of the submariners generally can take. Such an inquiry will be the subject of a successive report.

2) The acute bronchial catarrh was observed only in three subjects and in a rather slight form, appearing about a week after the inhalation of the gas.

Its symptoms were characteristically coughing, a constriction in the thorax, and light expectoration. Objectively, whistles, hisses, and rhonchi were present in the entire pulmonary region. Some wheezing with large blisters was present. In one case, there was a slight temperature elevation (37.2--37.5) for four days.

In so far as the duration of the diseased form is concerned, in one case recovery occurred in about 15 days; in a second case in about 30 days; in the third case, light, mature catarrh still remained which, however, showed remarkable improvement 40 days after its appearance.

3) The headaches, the retrosternal pain, and the yellowish-green coating on the tongue occurred in a majority of those stricken. Generally, these symptoms disappeared about 10 days from their onset.

Records of Chlorine Gas Poisoning in General

The Properties of Chlorine

Under ordinary conditions of temperature and pressure, chlorine is a greenish-yellow gas with a characteristic odor. It irritates the respiratory organs at a concentration of 3.6 parts per million of air (10 mmgr. per m^3). The maximum limit that a normal man can stand for 1 min without damage is 100 mmgr. per m^3. The Haber mortality product, or lethal index, is the product of the concentration of chlorine in the air expressed in mmgr. per m^3, [multiplied] by the duration of its action expressed in minutes to the point that death occurs is 7500. It diffuses throughout the atmosphere at great speed.

The process of the action of chlorine on the human organism has given rise to many valid hypotheses and theories; one of the most trustworthy is that hydrochlorous acid and hydrochloric acid (caustic) form from the chlorine, and the toxicity consists of the obstruction of the imbibition of water by the tissues (dessication).

Pathologic Anatomy

Respiratory apparatus -- In the cases of light poisoning, the pulmonary capillaries dilate, causing a blood stasis and an inflamation that does not

go beyond the large and medium bronchi, provoking an acute bronchial catarrh that represents the first true lesion from chlorine. Through longer exposures or high concentrations, the lesions extend to the cells of the alveolar epithelium, deepen into the pulmonary parenchyma, inducing the onset of particular alterations that principally lead to the formation of an acute pulmonary edema, very serious itself or, secondarily because of cardiac insufficiency, a cause of death.

An unusual fact is the nonuniformity of the distribution of the alveolar alterations. Besides a group of alveoli of normal appearance, full of air, we find others, dilated, containing pure edematous liquid; others contain corpusculated liquid with leucocytes, red corpuscles, desquamated alveolar cells; other groups, especially near the pulmonary edges, are strongly emphysematous and, finally, atelectasis zones. The unusual and curious distribution of the various lesions caused by a poison uniformly distributed in the air is explained by the hypothesis that the irritation of the nerve endings of the bronchioli are caused by spastic contractions of the wall, spasms that here and there obstruct the afflux of the poison in the respective alveolar zone.

Serious alterations take place especially at the expense of the pulmonary capillaries, particularly of their endothelium. By means of these alterations, the capillaries become permeable to blood plasma and red corpuscles, tending to give rise to hemorrhages.

Circulatory organs -- The heart appears to be enlarged by a notable straining by the right side. The dilatation of the right side of the heart is evidently tied to the resistance caused in the pulmonary circle because of the serious alterations in the respiratory organs. The general toxic factor also aids such a dilatation; in fact, with a microscopic examination,

one can verify characteristic alterations of a true, primary degenerative process taking place at the expense of the cardiac muscular fibers, consisting for the most part of turbid swelling and fat degeneration.

In the cases of delayed death, there is a tendency toward the formation of thrombi. Frequently, they are emboli in the arteries of the lower limbs, in the coronary arteries with the formation of a myocardial infarction, and infarctions in the lungs and kidneys.

There is almost always an increase in the red blood corpuscles, to the detriment of the blood, an increase in the viscosity, and in the hemoglobin rate, that can reach 140 in serious cases. There is also hyperleucocytism.

In the digestive organs, the alterations are much less serious and are generally of the second order. In the stomach, intense hyperemia of the mucous membrane, small hemorrhages and sometimes true ulcerations are present. In the intestine, there is little or nothing to observe.

Among the parenchymatical organs, the liver is one of the worst victims. To a macroscopic exam, it usually appears enlarged, hard, and easily friable; the form is unchanged, the surface is smooth, shiny and the connective capsule strained. The color is dark red. On the surface of the section, the acinaceous structure is well-delineated. In the middle of the well-preserved hepatic parenchyma, there stood out, under microscopic examination, a part of a lobule or several lobules, the center of the progressive degeneration that reached the necrobiosis of the hepatic cell.

The spleen is enlarged, stumpy with capsule strained and smooth.

The kidneys are enlarged, of grayish color, and the crown is strained. After sectioning the kidney, one notes an increase in the thickness of the cortical substance. There is so much in the medullar substance that in the cortex there are visible, at times to the naked eye, small hemorrhage centers.

The histological exam confirms that it is a question of an acute hemorrhagic parenchymatical nephritis.

Nervous system -- The meninges appear intensely hyperemic with parvicellular infiltration, especially around the blood vessels. Hyperemia and small hemorrhages are found both in the gray matter and, more particularly, in the white matter. At the histological exam, the nerve cells exhibit the so-called primary poisoning of of Nissl (tumefaction, central hydrolysis, transmigration of the nucleus to the periphery with the appearance of little vacuoles in the cell body and transmigration of the chromatic mass).

Symptomology

On the basis of the intensity of the poisoning, which is tied to the greater or lesser concentration of the poison at the time of exposure and to the quantity absorbed, the authors distinguish between three clinical forms:

a) The most serious form; very rare, for massive fulminating poisoning. The victim, after a few disorderly and unseemly breaths, falls as if struck by lightning, almost always because of very acute pulmonary edema.

b) The serious form and the moderately serious form: they are the ones most commonly encountered. In their development, three successive periods can be distinguished:

1) Irritating period, characterized by irritation of mucous membranes, difficulty breathing, constriction in the throat and psychic depression. Later, deep asthenia and an irritable cough appear.

2) Remission period -- the distress and the cough cease and there remains a light retrosternal pain which is aroused during the respiratory act, and some level of asthenia for a time more or less as long as the convalescence. This comes in the moderately serious form.

3) Period of state or asphyxiated period: this period belongs to the serious forms, and one passes to it directly from the irritated period. The dominant fact is the pulmonary edema with all of its symptoms, accompanied by tachycardia (120-200), generally low arterial pressure (Mx 80-110), liver and spleen enlargement on palpation, slight jaundice, oliguresis, oscuria, ischuria, albuminuria, serious psychic depression, intense headaches, drowsiness, and fever (38-39).

c) Light form. The symptoms described above can subsist, either totally or in part, but in a more attenuated form. On the basis of the group of symptoms and of the lesions presented, three types can be distinguished.

1) A pulmonary type with bronchiti and cough, generally epirectic;

2) A gastrointestinal type with nausea, vomiting, abdominal pain, constipation, and coated tongue;

3) A nervous type with headaches, pains in the rhachis, physical and psychic asthenia.

Comparing the preceding symptomology of chlorine poisoning extracted from the literature, and the symptoms that I found in the personnel of the submarine, I can conclude that in my case it is a question of poisoning that is in between the moderately serious form and the light form; in fact, in the said personnel, one can find all types and characteristics of the light form and the moderately serious form.

Courses and Complications

The most serious forms are quickly fatal; in the serious forms and moderately serious forms, the lethal result generally occurs within the first three days; after this period, the fatality is difficult to determine. Light forms move rapidly toward recovery and the victims with moderately serious forms can return to their normal activities within 2 weeks.

After the acute period, one often has persisting frontal headaches, epigastric pains, light bronchitis, and a protracted inability not only for intense muscular work, but also for moderate work. Spasmodic fits of nocturnal bronchial asthma are often easily observed.

Frequent microbiocide complications: bronchitis, bronchial pneumonia, massive pneumonia, purulent pleurisy. Pulmonary emphysema is also a frequent occurrence.

Aftereffects

The most important are those detrimental to the respiratory organs:

a) To the detriment of the larynx, hypotrophy and hypotonicity of the vocal cords,

b) Simple, recurring bronchitis,

c) Bronchitis with emphysema

d) The recurring edematous congestion; not very frequent,

e) Pulmonary sclerosis, like bronchitis with emphysema, is most frequent,

f) Pulmonary tuberculosis is not a very frequent aftereffect, but it is necessary to point out that the poisoning certainly carries on an activity that is not negligible in the development of tuberculosis.

Prognosis

This is based on all of the symptomatological and clinical elements reviewed: the quantity of the chemical element inhaled, the immediacy or delay of first aid, the individual's resistance, the behavior of the heart, the kidneys and the arising of complications. The persistance of tachycardia, hyperteremia, arterial hypertension, and the level of pulmonary reaction are inauspicious prognostic signs. In general, the prognosis is always inauspicious in the most serious forms; in the serious form, the prognosis is reserved, and it is good in the light form.

First Aid and Therapy

a) Suppress the action of the poison: this is done by removing the victim from the poisoned atmosphere immediately, carrying him into a pure atmosphere or applying a gas mask;

b) Clear the organism of the poison:

1) Absolute rest, avoiding every movement in order to reduce the oxygen requirements to a minimum;

2) Heat: avoid the dispersion of muscular energy and consequently limit oxygen requirements;

3) Administration of oxygen. This is the ideal first aid. It must be administered in a pure form, not mixed with carbon dioxide. The quantity administered (10'-15' per h) must not be less than an average of 6 liters per minute for many consecutive hours (6-12 h);

4) Bleeding, only in the cases of red or blue cyanosis, with strong pulse and arterial hypertension. It must never be used in asphyxia or pallid cyanosis. In this last case, diaphoretic treatment is adopted. The bleeding must be early and abundant (from 400-500 cc);

5) Cardiotonics: caffeine, canfidrol [sic], strophantin; contraindicated: digitalis because of the depressing action on the heart; adrenalin, which, by its vasoconstricting properties, favors the development of acute pulmonary edema. Artificial respiration is dangerous because of the disabled anatomical condition of the lungs;

c) Treat the lesion suitably. In place of treatment, integrate those first aid measures described above with subcutaneous injections of emetine hydrochloride to provoke vomiting and with intravenous injections of calcium 10% to render the pulmonary alveoli less permeable to the blood plasma and to create an anti-inflammatory and decongestant action.

The stomach pains will be dulled with small doses of bicarbonate of soda at 3%. The expectorants must not be given during the period of greatest severity. The treatment will be completed by collutories at 5% of sodium bicarbonate and the introduction into the nostrils of some drops of gomenol oil both to soothe the irritation of the throat, of the larynx, and of the respiratory passages, and to prevent facile and possible complications.

The diet must be liquid and administered in small quantities for the first 5-6 days: water, coffee, tea, milk, preferably tepid. Slowly, one can pass to a more strengthening diet, but always lacto-vegetarian.

In this article, I have stated the results acquired by the personal observations of a case of cumulative chlorine gas poisoning on board a submarine. I believe it useful to note, since it is a chapter of remarkable interest in the pathology of submariners, that it merits being well-known for its importance, especially insofar as it regards the complications and the aftereffects of the serious cases.

Summary

The author describes a case of cumulative chlorine gas poisoning that happened on board a submarine by seepage of seawater into the storage batteries making several clinical observations about the symptoms of major interest.

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